

REMARKSRejection of the Claims under 35 USC 102:

Claims 1, 3, and 7 have been rejected under 35 U.S.C. 102(b) as being anticipated by Sahu et al. 1996. Applicants respectfully disagree. The peptide isolated by Sahu et al binds to human C3 protein and prevents its activation, thereby blocking complement activation in human blood. It is noted by Sahu et al. that this peptide is effective in inhibiting (by 50%) the alternative pathway of complement activation at a concentration that is two-fold greater than the concentration of C3 in serum (page 889, second column, first paragraph). Inhibiting the classical pathway required even higher levels of peptide. Therefore, in order to be resistant to inactivation in blood, the phage taught by Sahu et al must be present at a concentration that is more than two-fold greater than the concentration of C3 in the blood. Furthermore, it is noted that Sahu et al. do not actually show that the phage displaying their peptide actually inhibits complement activation. While C3 binding is shown for the phage clone 9, Complement pathway inactivation is only shown for the isolated peptide.

Applicants respectfully disagree with the action's argument that a phage which can bind to and inactivate a finite number of C3 molecules must itself be resistant to serum inactivation. First, Sahu et al. do not show that M13 phage displaying their peptide actually inhibits complement activation. Second, as addressed in the Applicants specification, page 19 line 27 to page 20 line 20, a number of different proteins may bind to viral or non-viral particles resulting in clearance or inactivation of the particle. In fact, CRP can activate complement when bound to phosphocholine and galactosyl residues within liposomes, but when bound to T7 phage protects the phage from complement-mediated inactivation.

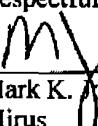
The action states that it is the "applicants' own observation that inhibition of complement activation by binding to CRP prolongs the half-life in the blood for phage bearing the CRP-binding polypeptides." Applicants respectfully disagree. Applicants have neither observed nor claimed that binding of CRP by the phage inhibits compliment activation pathways in blood. Rather, it is the Applicants' observation that binding of CRP by T7 phage displaying the appropriate peptide, prevents inactivation of the phage by complement activation pathways. The complement pathways may still be active in the blood, but are ineffective in inactivating the phage.

Rejection of the Claims under 35 USC 103:

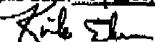
Claims 4 has been rejected under 35 U.S.C. 103(a) as being unpatentable over Sahu et al. 1996 in view of the 1997 Novagen Catalog. Applicants respectfully disagree for the reasons stated in response to the 102(b) rejection.

The Examiner's rejections are now believed to be overcome by this response to the Office Action. In view of Applicants' amendment and arguments, it is submitted that claims 1, 3, 4 and 7 should be allowable. Applicants respectfully request a timely Notice of Allowance be issued in the case.

Respectfully submitted,


Mark K. Johnson Reg. No. 35,909
Mirus
505 South Rosa Road
Madison, WI 53719
608-238-4400

I hereby certify that this correspondence is being facsimile transmitted to the USPTO or deposited with the United States Postal Service with sufficient postage as express mail in an envelope addressed to: Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450 on this date: 7/22/04



Kirk Ekena